https://doi.org/10.17816/ecogen14530

谷类作物抗白粉病的遗传多样性

GENETIC DIVERSITY OF CEREAL CROPS FOR POWDERY MILDEW RESISTANCE

© E.E. Radchenko, R.A. Abdullaev, I.N. Anisimova

Federal Research Center "N.I. Vavilov All-Russian Institute of Plant Genetic Resources", St. Petersburg, Russia

Cite this article as: Radchenko EE, Abdullaev RA, Anisimova IN. Genetic diversity of cereal crops for powdery mildew resistance. *Ecological genetics*. 2020;18(1):59-78. https://doi.org/10.17816/ecogen14530.

Received: *28.06.2019* Revised: *12.11.2019* Accepted: *19.03.2020*

✧ *Blumeria graminis* 病原体）是最常见的和有害的真菌病害的谷类作物，特别是在潮湿的气候地区。病原体的特征是与寄主植物的基因型有不同的相互作用。防治白粉病最合理、最便宜、最环保的方法是培育受不同抗性基因保护禾谷类作物的品种。通过对栽培植物遗传资源的收集、野生亲缘抗性的渗入等方面的研究，可以补充有效基因的存量，以及通过使用传统（人工诱变）和生物技术方法，包括基因组编辑创造的突变体。在这方面，近几十年来，发现了和识别了抗性基因的兴趣增加，找出其结构和功能组织，以及性状形成的分子机制分析。该研究综述了小麦、大麦、燕麦等主要谷类作物抗白粉病确定基因当前信息的进展。给出了在分子水平上鉴定的小麦和大麦基因的列表。其中：编码NLR和CLR蛋白的基因（软质小麦的 *Pm2*、*Pm3*、*TaMla2*、*TaMla3*、黑麦的 *Pm8*、大麦的 *Mla*）、受体样蛋白（大麦的 *Mla*）、转运蛋白和受体样激酶（小麦的 *Lr34*、*Lr67*、*Pm21*）。

✧ **关键词：** *Blumeria graminis*；寄生物—寄主植物的互动；阻力；R—基因；蛋白质；组织结构的功能

❀ Powdery mildew (causal agent *Blumeria graminis*) is a widespread and harmful fungi disease of cereal crops especially in the regions with humid climate. The pathogen is differentially interacting with plant host genotypes. Growing cereal crop varieties protected with different resistance genes is the most rational, costly and ecologically safe way of combating powdery mildew. The supply of effective genes can be increased due to studies of crop genetic resources collection, introgression of resistance from wild relatives, and also at the expense of mutant forms created with the use of traditional (induced mutagenesis) and biotechnological methods including genome editing. This causes the increasing interest to searching and identifying resistance genes, elucidation of their structural and functional organization, and analysis of molecular mechanisms of the character development. The review summarizes modern information on the identified genes of powdery mildew resistance of the main cereal crops – wheat, barley and oat. The list of wheat and barley genes identified at the molecular level is presented. It includes genes encoding NLR and CNL proteins (*Pm2*, *Pm3*, *TaMla2*, *TaMla3* genes of wheat, rye *Pm8* gene, barley *Mla* gene), receptor-like proteins (barley *Mlo* gene), transport proteins and receptor-like kinases (*Lr34*, *Lr67*, *Pm21* of wheat).

❀ **Keywords:** cereals; *Blumeria graminis*; parasite–plant host interaction; resistance; R-genes; proteins; structural and functional organization.

引起禾谷类作物白粉病的病原体是专性寄生菌*Blumeria graminis DC*。该物种包括许多形态相似的形态，其与寄主植物的特化程度不同[1]。几种专门形式发育在谷类作物上：*f. sp. tritici Marchal* (在*Triticum L.* 属的品种上，以及在*Aegilops L.*及其他野生谷物)，*f. sp. hordei Marchal* (在*Hordeum L.*属的品种上)，*f. sp. secalis Marchal* (在*Secale L.*属的植物品种上) 和*f. sp. avenae Marchal* (在*Avena L.*属的品种上)。直到2001年，白粉病无在黑小麦被发现料。在欧洲这种栽培上的疾病传播是由于一种新的*forma specialis triticosecale*的出现[2, 3]。

植物抗病性是限制白粉病危害的主要原因之一。抗性基因型的植物选择对抗这种疾病是一种根治，同时也是最便宜、最环保的方法。遗憾的是，该病原体的特征是与寄主植物的基因型有差异的相互作用[4]。这意味着栽培品种在抗白粉病基因上的一致性为真菌的适应性微进化创造了条件。

*B. graminis*与植物的相互作用受一个《基因对基因》关系支配[5]：每个宿主抗性基因对应一个特定的寄生菌毒力基因。寄生菌致病基因

的突变会导致宿主的抗性基因失效。抗性基因通常是显性的，因其进化年龄更大，而且寄生菌（熟悉的伴侣）的毒性是由隐性基因控制的。人们认为，抗性和毒力具有《正》作用（相互作用的基因产物），而易感性和毒力具有《负》作用[6]。

同一品种可能对不同种群的病原菌表现出不同的抗性基因。抗性基因表现的稳定性不同，取决于周围和遗传环境。在萌发阶段出现的抗性基因（《原生基因》）通常在植物的整个生命周期中都是活跃的。抗性表达在植物个体发生过程中可能发生变化。

寄主植物对病原菌的抗性通常与植物的超敏反应有关。这是一种植物的保护反应，表现为当有害生物侵入时，局部细胞迅速死亡，并伴随着有毒物质在死亡细胞中积累。植物病原体与植物的相互作用包括以下几个阶段：诱导子（激发子）的分离、由植物细胞利用受体识别激发子、向基因组传递信号、激活免疫应答基因转录、保护化合物的合成[7]。

为了预防白粉病植物列病的发生，需要培育具有不同抗性基因的品种。通过研究栽培植物的收集、野生亲本抗性基因的导入以及利用传统和生物技术方法创造的突变体，可以补充有效基因的存量。目前，基因渗入在扩大多种多样禾谷类作物通过抗性基因对于*B. graminis*具有特有的意义。对小麦和大麦抗白粉病的遗传性进行了深入的研究。然而，关于位点的遗传结构及其编码产物的信息仅为少数基因所知的。

本文目的是总结有关抗白粉病基因多态性的已知的文献资料。

控制谷类作物对白粉病抗性的基因

目前，在62个控制小麦白粉病抗性的位点（*Pm1—Pm65*）中发现了92个等位基因（见表1）。大多数基因是显性的，并在植物个体发育过程中表达。其中它们有44个等位基因：就是*Triticum aestivum L.*，26个从不同*Triticum*种传播的，11个从*Aegilops spp.*，5个从*Secale cereale L.*，6个基因从*Dasypyrum villosum (L.)* Borbás（*Haynaldia villosa (L.) Schur*的同义词）、*Thinopyrum ponticum (Podp.)* Z.W. Lin & R.C. Wang、*Thinopyrum intermedium*(Host) Barkworth & D.R. Dewey和*Agropryron cristatum* (L.) Gaertn基因渗入的。20多个抗性基因被给予了暂时的符号。

除了表型表现明显的基因外，小基因(quantitative trait loci—QTL)也能控制相当高水平的对真菌的抗性（主要与年龄有关，表现在旗叶阶段）。小麦在21条染色体上至少有119个QTL的株龄相关抗性。结果表明，*Lr34/Yr18/Pm38/Sr57*（7DS染色体）、Lr46/Yr29/Pm39/Sr58 (*1BL*)和*Lr67/Yr46/Pm46/Sr55* (4DL)为成体植株对褐、黄、茎锈病和白粉病提供长期抗性[84]。

据认为，由于编码序列结构的差异，与受体物种的基因相比，基因渗入提供了更大范围的长期稳定性。因此，在中国受*Pm21*基因保护的*D. villosum*（易位T6AL.6VS）品种尽管经过了广泛的栽培，但在40多年的时间里仍然对病原体具有抗性。目前，人们对来自中间偃麦草的*Pm40*抗性基因的新型育种材料寄予厚望[85]。然而，无论是《自己》抗性基因还是基因渗入到软质小麦基因组的抗性基因都有不同的效率和《使用寿命》，此外，该菌还能克服具有外源基因的品种和亲缘关系较近的品种的抗性。例如，*Pm8*基因育种的广泛使用自1970年代（易位T1BL.1RS）和后续培养基因同类品种的大面积导致在20世纪90年代初，欧洲种群中该真菌的毒力*Pm8*无性系的含量达到了100% [86]。在某些情况下，稳定性的迅速丧失可以用*Pm8*基因的抑制来解释。因此，在瞬时表达的实验中，证实了来自Petkus黑麦品种*Pm8*抗性基因的小麦细胞系的表皮细胞中，存在*Pm3*小麦基因的功能性和非功能性等位基因时，对抗性反应的抑

制[87]。不幸的是，目前确定长期抗性的唯一可靠标准是培育抗性品种的经验。

目前已知的控制大麦抗白粉病有100多个基因，其中大部分是*Mla*和*Mlo*基因的等位变异。它们出现于各种来源的样本中，大部分来自以色列。已描述了*Mla*基因（1H染色体）的39个等位基因[88-90]和*Mlo*基因（4H染色体）的约40个等位基因[91]。遗憾的是，大多数等位基因对病原体无效。几乎全世界大麦品种对病原体的长期抗性是由*mlo11*基因和在某种程度上由*mlo9*基因来提供的。目前，欧洲75%的春大麦品种受这些基因的保护[92]。

在整个植物个体发育过程中，鉴定了11个控制燕麦抗性对*B. graminis (DC.)* E.O. Speer *f. sp.* Avenae Em. Marchal的基因[93]。Jumbo品种受显性*Pm1*基因的保护，其定位在一条1C染色体上[94]。来自*Avena sterilis L. var. ludoviciana*转入培养燕麦（*Mostyn*品种）的显

表1

小麦抗白粉病基因

染色体	*T. aestivum*抗性基因	来自相关物种的抗性基因
1A	*Pm3a*, *Pm3b*, *Pm3c* [8, 9], *Pm3d*, *Pm3e*, *Pm3f* [10], *Pm3g* [11], *Pm3i*, *Pm3j* [12], *Pm3l* [13], *Pm3m*, *Pm3n*, *Pm3o*, *Pm3p*, *Pm3q*, *Pm3r* [14]	*Pm3h* (*T. durum*) [12], *Pm3k* (*T. dicoccum*) [13], *Pm25* (*T. boeoticum*) [15], *Pm17* (*S. cereale*) [16, 17]
2A	*Pm4c* (*Pm23*) [18], *Pm65* [19]	*Pm50* (*T. dicoccum*) [20], *Pm4a* (*T. dicoccum*), *Pm4b* (*T. persicum*) [21], *Pm4d* (*T. monococcum*) [22]
3A	*Pm44* [23]	–
4A	*Pm61* [24]	*Pm16* (*T. dicoccoides*) [25]
5A	–	*Pm55* (*D. villosum*) [26]
6A	–	*Pm56* (*S. cereale*) [27], *Pm21* (*Pm31*) (*D. villosum*) [28]
7A	*Pm1a* [29], *Pm1e* (*Pm22*) [30], *Pm9* [31], *Pm59* [32]	*Pm1b*, *Pm1c* (*Pm18*) (*T. monococcum*), *Pm1d* (*T. spelta*) [29], *Pm37* (*T. timopheevii*) [33], *Pm60* (*T. urartu*) [34]
1B	*Pm28* [35], *Pm39* [36]	*Pm32* (*Ae. speltoides*) [37], *Pm8* (*S. cereale*) [38]
2B	*Pm52* [39], *Pm63* [40]	*Pm6* (*T. timopheevii*) [41], *Pm26* (*T. dicoccoides*) [42], *Pm33* (*T. persicum*) [43], *Pm42* (*T. dicoccoides*) [44], *Pm49* (*T. dicoccum*) [45], *Pm64* (*T. dicoccoides*) [46], *Pm57* (*Ae. searsii*) [47], *Pm51* (*Th. ponticum*) [48], *Pm62* (*D. villosum*) [49]
3B	–	*Pm41* (*T. dicoccoides*) [50], *Pm13* (*Ae. longissima*) [51]
4B	–	*Pm7* (*S. cereale*) [52]
5B	–	*Pm30* (*T. dicoccoides*) [53], *Pm36* (*T. dicoccoides*) [54], *Pm53* (*Ae. speltoides*) [55]
6B	*Pm11* [56], *Pm14* [57], *Pm54* [58]	*Pm27* (*T. timopheevii*) [59], *Pm12* (*Ae. speltoides*) [60], *Pm20* (*S. cereale*) [61]
7B	*Pm5b*, *Pm5d* [62], *Pm5e* [63], *Pm47* [64]	*Pm5a* (*T. dicoccum*), *Pm5c* (*T. sphaerococcum*) [62], *Pm40* (*Th. intermedium*) [65]
1D	*Pm10* [66], *Pm24a* [67, 68], *Pm24b* [69]	–
2D	–	*Pm58* (*Ae. tauschii*) [70], *Pm43* (*Th. intermedium*) [71]
4D	*Pm46* [72]	–
5D	*Pm2c* [73], *Pm48* [74, 75]	*Pm2a* (*Ae. tauschii*) [76], *Pm34* (*Ae. tauschii*) [77], *Pm35* (*Ae. tauschii*) [78], *Pm2b* (*A. cristatum*) [79]
6D	*Pm45* [80]	–
7D	*Pm15* [57], *Pm38* [81]	*Pm19* (*Ae. tauschii*) [82], *Pm29* (*Ae. ovata*) [83]

性*Pm3*基因定位在一条17A染色体上[94–96]。除了*Pm3*外，Rollo品种还在4C染色体上有第二个显性抗性基因*Pm8*[94]。对渐渗系病原体Cc6490的抗性，在*A. barbata*的参与下接收，由定位在一条18D染色体上的*Pm4*基因控制。*Pm5*抗性基因（19A号染色体）是从*A. macrostahya*中种质渗入的[94,97,98]。在Bruno品种中发现定位于10D染色体上的*Pm6*隐性抗性基因。APR122繁育行，其谱系有*A. eriantha*，受定位在一条13A染色体上*Pm7*显性基因的保护。从*Avena hirula*转入培养燕麦的*Pm2*基因的定位仍不清楚[94]。*A.byzantina* AVE2406和AVE2925样品各携带一个有效显性抗性基因：分别*Pm9*（*16A*染色体）和*Pm10*（10D染色体）[99]。有效抗性基因*Pm11*在CN113536（*A. sterilis*）样品中得到鉴定[93]。

耐药基因*Pm1*、*Pm3*和*Pm6*的供体在许多欧洲国家的育种项目中被广泛使用[100–102]，受到病原体的强烈影响。最高抗性水平是由*Pm4*基因提供的，而在欧洲*Pm7*基因的效果稍差[103]。*Pm4*基因的标记已经被开发出来，其适合于标记介导的选择[104]。

对大量*A. sativa*材料的研究结果表明，有效的抗白粉病基因的培养多样性较低[95,100–102]。来自波兰的一个Canyon品种被分离出来，其可能受到一种新的抵抗病原体的基因组（基因）的保护[102,103]。在六倍体裸燕麦品种中，抗病性的来源非常少。因此，在350份被研究的*A. sterilis*样本中，只有10份是抗病性的[105]。在不同类型的*A. sterilis*中，最有趣的样品是CN67383和CN113536，其有新的抗性基因[106]。研究表明，四倍体*A. magna*和*A. murphyi*品种的样本是有效的抗病供体，此外，所有被选择的形式都来自地中海国家（摩洛哥和西班牙）[107,108]。

在燕麦中，与年龄有关的植物对白粉病的抗性也是众所周知的。因此，鉴定出了9个地区性品种和2个具有高水平的成年植物抗性的商业品种[109]。

控制谷类作物抗白粉病基因的结构和功能多样性

在细胞水平上，植物有两条防御病原体的防线：外层和内层。外层保护由位于细胞表面的横跨膜模式诊断受体（pattern recognition receptors — PRR）提供的，其能够识别保守的病原相关分子结构（模式）（pathogenassociated molecular patterns — PAMP），如脂多糖、肽聚糖和细菌蛋白。主要的横跨膜受体是受体样激酶（receptorlike kinases — RLK）和受体样蛋白（receptorlike proteins — RLP）。内层防线由细胞质受体提供，其中大部分（由耐药基因或*R*—基因编码的）属于NLR蛋白的保守家族，其特征是存在核苷酸结合位点（nucleotide binding site — NBS）和富含亮氨酸（leucine rich repeat — LRR）的结构域。效应蛋白可以被NLR细胞的受体直接识别，或者间接地通过变体与宿主NLR蛋白的结合[110–113]。

R—基因的分子鉴定研究采用定位克隆、比较基因组学和突变基因组学等方法[114]。然而，禾谷抗性基因的克隆和测序数量仍然较少，且主要局限于小麦和大麦。

编码NLR型受体的*R*—基因通常属于多基因家族。它们的特征是由于节段性重复、重组、不等交换、点突变和发散性选择具有基因组中的簇群组织和高度的可变性[115]。描述了一系列*R*—基因的多重等位基因，特别是小麦*Pm3*[12]和*Mla*的基因[116,117]，还有大麦的基因[90]。

据认为，与抗性基因相比，效应基因具有更高的变异水平。这尤其适用于小麦和大麦白粉病病原体证明的，其毒力效应物比许多其他基因进化得更快，这使得病原体能够克服相关*NLR*基因的影响[118]。保护反应是各种基因、蛋白质和调节分子相互作用的结果。在一个形式化的形式中，小麦中这些相互作用的图像以一个重建的基因网络的形式呈现出来，其描述了与致病真菌免疫反应形成相关的功能基因群[119]。

迄今为止，在小麦分子水平上已经鉴定出9个控制小麦抗白粉病的*R*—基因。其中克隆了和测序了大麦一个基因和黑麦一个基因。小麦的*Pm2*、*Pm3*和*Pm60*基因、大麦的*Mla*基因和黑麦的*Pm8*基因编码属于NLR—受体家族的蛋白质。长期的抗性是由编码具有激酶或转运功能的蛋白的基因决定的：从*Haynaldia villosa Pm21*种质渗入的，以及具有多效性的位点*Lr34/Yr18/Sr57/Pm38*和*Lr67/Yr46/Sr55/Pm46*。MLO蛋白是*Mlo*位点（免疫应答的负调节因子）的产物，其生化功能尚未完全阐明[120-132]（表2）。

对*Lr34/Yr18/Pm38/Sr57*和*Lr67/Yr46/Pm46/Sr55*位点的基因进行了测序，同时对褐、黄、茎锈病、白粉病等几种病原菌具有抗老化能力。已经表明，*Lr34/Yr18/Pm38/Sr57*基因携带者对真菌的多重抗性，以及叶尖端坏死（*Ltn1*标记）是由*Lr34*基因的影响，其定位于7D染色体的短臂上*Xgwm295*位点附近，并与*Yr18*、*Pm38*、*Sr57*基因相同[124]。*Lr34*基因的产物属于ABCG类ATP结合盒转运蛋白（ATP binding cassette — ABC），包括1401 a. o.以及两个胞质核甘酸结合域和两个疏水跨膜结构域。敏感而抗性基因型的*Lr34*等位基因在两个多态性位点上存在差异，其改变其中一个横跨膜域的结构[124]。*Lr34*基因参与质膜重构，并伴随着细胞内磷脂酸的积累以及磷脂酰丝氨酸移动水平的升高。在*Lr34*基因控制下，磷脂的再分配对膜蛋白的组成有影响，还激活了对应激因子的响应，这有助于大麦*Lr34*转基因植株中性脂质的积累[133]。

具有多效性*Lr67*基因的产物，设想的是STP13 H^+类同向转运单糖的已糖载体，其尺寸是514 a. o.，含有12个跨膜螺旋，可通过细胞膜传递葡萄糖。抗性型（*Lr67res*）和敏感型（*Lr67sus*）基因型的蛋白仅在两个氨基酸残基上存在差异，保守的STP样己糖植物转运。Lr67sus蛋白和相关的同源等位基因编码的蛋白起高亲和力的葡萄糖转运蛋白的作用。同时，*Lr67res*等位基因具有负主导作用。Lr67res蛋白与这些同位等位基因的产物相互作用形成异二聚体，这导致导致葡萄糖摄取减少以及减缓病原真菌的生长[125]。实验证实了*Lr67*基因所确定的抗性机制的保守性：小麦抗性等位基因*Lr67res*测定了转基因大麦植株对矮秆锈病和白粉病的抗性，并增强了致病相关基因*PR1*、*PR2*、*PR3*的表达。但与小麦不同的是，在苗期就出现了抗性，这显然是由于该基因在不同遗传背景下表达水平的差异造成的[134]。

分别在同线区域定位于小麦1AS染色体上和黑麦1RS染色体上的*Pm3*和*Pm8*基因是同源基因。鉴定的候选基因产物*Pm3b*（1415 a. o.）和*Pm8*（1375 a. o.）具有显著的相似性。它们的蛋白质序列有81%的氨基酸残基相同，并且大多数多态位点位于与胞质溶胶接触的相同的富含亮氨酸的重复序列中。不同Triticeae族的两个同源基因的序列代表了同一种单体型的复合体。这意味着这两个基因在750万年前小麦物种从一个共同祖先分散后独立进化了，但保留了一个共同的功能[122]。

*Mla*位点（*Mildew resistance locus A*）位于1H染色体的短臂上，由30多个等位基因组成，决定大麦对白粉病的种特异性抗性[89]。*Mla*基因的特征是具有异常高水平的功能多样性（对一组特定种族的特异性），编码NLR蛋白，包括超螺旋化（coiled coil — CC）、核苷酸结合和亮氨酸富结构域（coiled coil — nucleotide binding site — leucine rich repeat — CNL受体）。它们来自不同来源被种质渗入到了培养大麦的基因组，其中包括*H. spontaneum*野生品种。*Mla*介导的耐药的特点是超敏反应的迅速发展[135]。*Mla*等位基因呈现高度多态性。据说每个*Mla*等位基因都能识别编码*B. graminis*非病原性效应的*AVRa*基因。因此，通过对17个含有各种AVR_a基因的*B. graminis*分离物的转录组分析，识别出了编码假定效应子的AVR_{a1}和AVR_{a13}变体，其分别由*Mla1*和*Mla13*等位基因编码的大麦免疫受体识别[115]。许多研究人员已经获得了关于大麦*Mla*位点结构组织的数据。特别是发现了Morex品种的*Mla*

位点含有一簇CNL编码的基因，属于*R*—基因同系物（resistance genes homologues — *Rgh*）的三个相异的亚科。抗性由*Rgh1*亚家族的等位变异控制的[89, 128, 136]。这些结论通过对50个样本*H. spontaneum*的转录组研究得到了证实，这些样本代表了生长在新月沃土领土上的9个种群。*Mla*转录本的多样性与样品来源无关。然而，根据可介导细胞死亡的两个N-端超螺旋信号域的结构，所有识别的转录本被分为两个亚科，其中一种包括所有已知的决定对*B. graminis*抗性的MLA受体变体[137]。

通过生物信息学分析，大麦基因组中发现了175个*CNL*基因，属于3个系统发育组。大多数已鉴定的聚类位于染色体的额外中心体周围区，这决定了这组基因迅速分散所必需的高度重组[138]。

大麦和小麦基因组中存在的微RNA的miR9863族已被证明在启动大麦*Mla*基因诱导的免疫应答中发挥了关键作用。该族4位成员在*Nicotiana benthamiana Domin*细胞中进行异源表达实验，进行*Mla*转录本的差异分裂，并抑制

表2

禾谷类作物抗白粉病测序基因序列表

基因	蛋白质	种类，基因型	文学参考
小麦			
Pm2	NLR	*T. aestivum*、CI12632/8线 (来自Chancellor品种选择)	[120]
Pm3b	CNL	*T. aestivum*、本地Chul品种、Chul/8*Chancellor线	[121，122]
Pm21a (*Stpk-V*)	丝氨酸和苏氨酸蛋白激酶	*H. villosa*、*T. durum*二倍体— *H. villosa*来自*H. villosa T. aestivum*与*T6VS.6AL*易位*T. aestivum*线与额外*H. villosa*染色体	[123]
Lr34/Yr18/Sr57/Pm38	ABC—运输者	*T. aestivum*、Thatcher *Lr34*线、Avocet *Lr34*、Forno、Chinese Spring	[124]
Lr67/Yr46/Sr55/Pm46	己糖运输者	*T. aestivum*、Thatcher *Lr67*线	[125]
Pm60	NLR	*T. urartu*、来自黎巴嫩和土耳其的样本	[34]
TmMla1	相同	*T. monococcum*、DV92线	[126]
TaMla2	CNL	*T. aestivum*、TAM104R线与6BS.6RL易位	[127]
TaMla3	相同	*T. aestivum*、TAM104R线与BS.6RL易位	[127]
黑麦			
Pm8	CNL	*S. cereale*、来自Petcus品种的线	[122]
大麦			
Mla	CNL	*H. vulgare*、 Morex品种	[128]
Mlo (野生型)	钙调素结合蛋白	*H. vulgare*、 Ingrid品种	[129]
mlo1, *mlo3*, *mlo4*, *mlo5*, *mlo7*, *mlo8*, *mlo9*, *mlo10*, *mlo13*, *mlo17*, *mlo26*	相同	*H. vulgare*、 Haisa、 Maltera Heida、 Foma、Carlsberg II、Diamant、Plena品种的诱导突变体	[129]
mlo12, *mlo16*, *mlo27*, *mlo28*, *mlo29*, *mlo30*	»»	*H. vulgare*、野生型等位基因和和 Sultan 5 *Mlo*品种携带者的诱导突变体	[130]
mlo11	»»	*H. vulgare*、自发突变 一个来自埃塞俄比亚的本地大麦样本。*H. vulgare* var. *spontaneum* 线来自以色列，土耳其和伊朗	[131]
mlo11 (*cnv2*)	»»	*H. vulgare*, 自发突变，来自埃塞俄比亚 Eth295的样本	[132]

MLA1蛋白的合成。这种相互作用的特异性是由成熟的miR9863的单核苷酸多态性以及*Mla*序列的miR9863结合位点上的两个SNPs来确定的，这取决于该位点的所有等位基因被分配到三个组[139]。

*Mla*位点基因与*Hor1*和*Hor2*基因相关，这两个基因控制着备用谷物蛋白的合成，即C和B大麦芽碱的合成[140]。有趣的是，小麦的*Pm3*位点与编码种子额外蛋白质，即低分子量谷蛋白亚基和额外麦胶蛋白的复杂*Glu3/-Gli1*-位点有关[122]。

在数百万年前发散的不同谷类属代表的基因组中发现了*Mla*基因的同源体。因此，在*T. monococcum*单倍体小麦基因组中发现了大麦*Mla*基因（*TmMla1*）的功能同源基因。大麦蛋白TmMLA1和HvMLA1的氨基酸序列具有78%相同的氨基酸残基。杂交蛋白TmMLA1的LRR结构域被HvMLA1蛋白的LLR结构域所取代的，被证明是功能性的，并对以前未知的*B. graminis*种确定了抗性[126]。在六倍体小麦中，发现了来自黑麦和*A. tauschii*的*Mla*基因*Sr33*和*Sr50*的同源基因，这两个基因对茎锈病（*Puccinia graminis f. sp. tritici*）提供了抗性[141,142]。克隆了和测序了*Mla* Triticum aestivum基因的*TaMla2*和*TaMla3*同源基因，其编码CNL蛋白，并在基因组中以多个拷贝为代表[127]。

大麦对*B. graminis*的非特异性长期抗性与位于4号染色体长臂上的*Mlo*位点（*Mildew locus O*）突变有关[143]。*Mlo*基因包括12个外显子，其编码的RLP蛋白分子量为60kDa，包含7个跨膜结构域和位于细胞内C—端上的钙调蛋白结合位点[129, 143]。野生型*Mlo*基因在植物的各种器官、组织和细胞类型中均有表达，在防止细胞过早死亡以及应对生物和非生物胁迫方面发挥重要作用。然而，在感染条件下，它们有一个负面的作用，因为MLO蛋白通过Ca^{2+}依赖与钙调蛋白的相互作用抑制对病原体渗透的保护反应，并防止过氧化氢对真菌渗透部位的表皮和叶肉的损害。因此，MLO蛋白通过抑制排斥反应来防止氧化爆炸和细胞死亡[144,145]。在隐性等位基因纯合的植物中，缺失MLO蛋白（功能缺失突变），并对*B. graminis*出现非特异性的性抗性。在另外两个基因*Ror1*和*Ror2*（对*mlo*抗性所必需的）的存在下，可以观察到完全的抗性[146]。在耐药的*mlo*突变体中，由于富含羟脯氨酸的糖蛋白的快速氧化交联，细胞壁在真菌穿透部位被重塑和加强[135]。*Mlo*突变体的特征是叶片损伤，这是表皮细胞壁并置（熟叶上的胼胝质沉积）后细胞过早死亡的症状的表现，即使在没有病原体的情况下也能观察到[147]。尽管许多限制负相关基因多效性的影响导致产量减少（例如，叶片过早枯萎）以及*Ramularia collocygni Sutton & Waller*真菌的*mlo*突变易感性，*mlo*等位基因在育种上的应用为中湿润气候地区的大麦*B. graminis*提供了稳定、长期的保护[148]。*Mlo*基因的突变导致功能显著的蛋白位点失活，以及终止密码子的出现。*Mlo*等位基因的特征是高频率的基因内重组，这导致相反的出现，即d野生型序列的还原[129]。

迄今为止，在*mlo*基因座中已经发现了40多个隐性功能缺失等位基因（*mlo*），其特征是具有不同程度的抗性：从部分（例如，通过化学诱变获得的*mlo12*和*mlo28*等位基因的特征）到完全（*mlo11*等位基因）。大多数突变是由单个氨基酸残基的替换引起的，较少是由缺失引起的。测定了若干等位基因的表型效应。因此，M.C. Kim等人[144]研究的14个突变体中有12个确定了对白粉病的长期抗性，并两个是通过减少与钙调蛋白的结合来降低敏感性。通过对单个等位基因序列的比较分析，发现了突变的聚类，即它们在某些外显子中的出现[130,131]。自发突变的*mlo11*最早于1930年在埃塞俄比亚采集的当地大麦样本中发现，并确定了对*B. graminis*所有种族的长期抗性，广泛分布于欧洲春季大麦品种中。抗性基因型的*mlo11*单倍型的特征是存在一个位于野生型*Mlo*等位基因序列之前的

11-12个重复单元的复杂的串联重复[131]。重复的情节包括5’调节序列的一段，其长度为3.5 t.p.o.，以及包含前5个外显子序列的1.1 t.p.o.编码区片段。来自这个序列的异常转录本破坏了*Mlo*转录本和野生型蛋白的积累，这显然决定了抗性。据认为，导致*mlo11*等位基因出现的突变是在大麦驯化后发生的[131]。*Mlo11*等位基因的另一种变体以重复次数的变化为特征—*mlo11* （*cnv2*），最近在埃塞俄比亚本地大麦的Eth295样本（*H. vulgare convar. deficiens var. nudideficiens*）中来自*Institute of Plant Genetics and Crop Plant Research*（*Gatersleben*，德国）的收藏品发现的[132]。*Mlo11*（*cnv2*）突变导致幼苗的部分抗性，而成年植株的完全抗性。该突变没有与细胞壁并置或坏死以及失去光合作用有关的负多效性效应。根据菌落数量和生长速率来估计相关的抗性被定义为定量的。在组织学水平上，标准等位基因*mlo11*和突变型等位基因mlo11携带者对真菌的抗性表现不同：在表皮细胞中，等位基因*mlo11*（*cnv2*）的基因型与真菌成功渗透区域的触，观察到细胞壁形成的并置，以及无出现坏死和叶肉细胞的崩溃。标准*mlo11*等位基因和突变型等位基因*mlo11*重复序列甲基化水平的差异与抗性表现的指标有相互的关系。*Mlo11*等位基因变体（*cnv2*）似乎源于祖先*mlo11*变体的自然选择，其结果是重复的元素与相邻区域的3‘端包含一个Stowaway类似的转座子的重组[132]。

基于*mlo*多态等位基因序列开发了分子标记[129,131]，其成功用于筛查育种材料[147]，并在采集样本中寻找突变等位基因的载体[149,150]。

*MLO*基因存在于植物和绿藻中。在高等植物中，包括谷类和双子叶植物，它们以多基因族为代表[148]。大麦*HvMlo*基因在软质小麦和软米水稻基因组中位于同一染色体上的位置。在软质小麦基因组中，*TaMloA1*、*TaMloB1*和*TaMloD1*同源物分别位于4BL、4DL和5AL染色体上。它们编码了三种相关的蛋白质，其中88%与大麦的ML0蛋白质相同，显然是由三个原始的小麦基因组演化而来。在软米水稻基因组中的*Mlo*直接同源，*OsMlo2*（偶联组3）在瞬时表达实验中恢复了大麦突变体对*B. graminis*的*mlo*敏感性[151]。在软米水稻基因组中发现了12个潜在的*MLO*基因族的代表[152]。为了确定它们的功能，作者结合了来自表达分析、转录组分析和系统发育分析的元数据。不同的*OsMLO*基因族成员有不同的组织特异性，参与不同的生理反应，包括对压力的反应。其中一个基因*OsMLO3*的表达下降时，*Magnaporthe oryzae*（T.T. Hebert） M.E. Barr稻瘟病的病原体受到影响，这表明该基因参与保护性反应[152]。

在*Brachypodium distachyon (L.) P.Beauv.*模型物种的基因组中发现11个保守型*BdMLO*基因，分布于5条染色体上。与其他植物一样，*BdMLO*基因包含7个保守的横跨膜区域以及和钙调蛋白结合位点。已鉴定的基因之一*BdMLO*可能是白粉病抗性的潜在候选基因[153]。在许多其他植物的基因组测序中，MLO同源物的数量从12个到19个不等[154]。单子叶植物和双子叶植物的*MLO*基因具有许多特定的特征，这些特征似乎是阴性选择的结果。然而，关于异种互补（一个物种的敏感性等位基因在另一个抗性基因型中的表达）的实验结果表明，在与植物白粉病病原体的相互作用中，存在许多保守的功能特征[154]。

讨论了获得*mlo*新变体的各种方法，包括使用RNA干扰抑制野生型等位基因（*Mlo*）表达，以及不使用转基因（TILLING）或有限使用的方法（使用TALEN和CRISP/CAS9基因组编辑系统）[148]。在J. Acevedo Garcia等人的研究中介绍了Cadenza软质小麦品种*TaMloA1*、*TaMloB1*和*TaMloD1*基因序列修改的TILLING技术的实际应用的结果[155]。作者获得了16个错义突变，每一个都导致一个氨基酸取代。在三倍和（在某些情况下）双突变体的基础上形成的细胞系具有对*B. graminis*抗性的特征，同时由于隐性*mlo*等位基因的阴性多效性作用而没有任何性状。

诱导突变的有效性依赖于其在*Mlo*基因中的位置：最有效的是影响膜蛋白第二和第三细胞质环的突变[156]。C.R. Ingvardsen等人[157]发现同源基因中诱导突变的有效性存在差异。利用TILLING技术，作者获得了Kronos品种硬粒小麦中*Mlo-A1*和*Mlo-B1*的一系列突变体。与*Mlo-A1*基因突变相比，*Mlo-B1*基因突变的影响一般较强，然而，在*Mlo-A1*和*Mlo-B1*两个位点中携带突变的基因型观察到最好的结果。

与植物防御反应有关的其他基因的突变可增加植物对白粉病的抗性。Y. Zhang等人[158]利用CRISP/CAS9技术获得了定位于1AS、1BL和1BL染色体上的*TaEDR1*（*enhanced disease resistance*）软质小麦保守基因的同源，其是抗性的负调控因子。三种*Taedr1*突变体对白粉病病菌具有抗性。

到目前为止，已知的40多个*mlo*突变等位基因中，只有两个：自发性*mlo11*和诱导性*mlo9*，在上世纪70年代和80年代早期用于大麦育种。目前，在中欧培育的半数以上的春大麦品种的免疫与*mlo*等位基因的使用有关[91]。

迄今为止所积累的关于抗性*B. graminis*的谷类遗传多样性特征的信息证实了N.I. Vavilov提出的《植物对传染病的自然免疫规律》的正确性[159]。已鉴定的栽培谷物对白粉病的主要抗性基因数量较多，且它们会随着时间不断更新。决定谷物种特异性抗性的基因有一个普遍的结构组织原则（属于免疫应答的NLR受体类），这使它们有可能与寄生虫的基因协同进化。所有这一切都与第一定律相一致，根据该定律，寄生虫的特化程度越高，就发现抗性形式的概率就越高。

《第二基本定律，其决定在一种特定的栽培植物中发现免疫品种和物种的概率，是否存在或不存在明显的遗传差异……免疫系统中最具反差的差异植物在细胞遗传学上已分化成不同的物种》[159]。该情况也说明了本文的数据。特别是，*Triticum*栽培品种有复杂基因组组成，具有多态性水平高的特性，而栽培大麦的遗传多样性水平相对较低。小麦在不同的染色体（主要是A和B基因组）中发现了大量的白粉病抗性基因，而大麦主要有两个具有大量等位基因的位点（*Mla*和*Mlo*）。

根据第三定律，免疫反应与植物的生态类型相对应，而在环境条件的对比中，免疫上最明显的差异是在对比鲜明的环境条件下发现的。N.I. Vavilov认为，免疫只在导致感染的条件下产生[159]。根据M.S. Wolfe和J.M. McDermott[160]，*B. graminis f. sp. hordei*的可能起源中心是地中海和中东。决定对*B. graminis*的种族特异性和长期抗性的*Mla*和*Mlo*基因的所有等位变异仅在东非和中东的样本中发现。

根据第四定律，在自然界中，群体免疫或复合免疫普遍存在[159]。有关抗性基因的结构和功能的数据使我们能够了解这种抗性的机制。与年龄有关抗性对几种病原体—小麦基因型的白粉病、褐、黄、茎锈病，携带*Lr34/Yr18/Pm38/Sr57*和*Lr67/Yr46/Pm46/Sr55*基因簇，实际上是由单一基因的多效性效应引起的，其编码蛋白，具有ABC—运输者转运功能（*Lr34*）和己糖运输者（*Lr67*）。

根据上述定律，N.I. Vavilov制定了第五和第六定律。《了解这种栽培植物的进化，〈……〉可以在很大程度上预测出育种者感兴趣的免疫形式的位置》。《生态地理正确性在免疫检测方面是比较普遍的，各种植物固有的，往往属于不同的属甚至科》[159]。这些模式是由先前讨论禾谷对病原体的抗性结果得到了确认。例如，在燕麦（*Avena*属）和大麦（*Hordeum*属）中，最抗性的形式来自地中海和北非[107,108]。

结论

谷物在抗白粉病方面具有广泛的遗传多样性。由于寄生虫与宿主关系的特殊性，许多基因很快就失去了效力，这就需要寻找新的

抗性供体。栽培种基因库具有相对贫乏抗性的形态。在这方面，来自野生亲缘的抗性的渗入最近已成为补充有效基因供应的最重要因素。因此，在迄今发现的92个软质小麦抗*B. graminis*的等位基因中，有48个传递的从野生亲缘的基因组：*Aegilops sp.*、*Secale sp.*、*Dasypyrum*（*Haynaldia sp.*）、*Thinopyrum sp.*、*Agropryron*。利用传统的诱变方法（例如，大麦的多个*mlo*等位基因），特别是利用TILLING和CRISP/CAS9技术对基因序列进行定向改变，可以获得新的抗性来源。抗性基因的结构和功能组织以及性状形成的分子机制等方面的信息还非常有限，只涉及小麦和大麦。从分子水平上鉴定的软质小麦的*Pm2*、*Pm3*、*TmMla1*基因、*T. urartu*野生一粒小麦的*Pm60*基因、黑麦的*Pm8*基因、大麦的*Mla*基因编码*NLR*和*CLR*蛋白；大麦的*Mlo*—受体蛋白；小麦的*Lr34*、*Lr67*、*Pm21*—转运蛋白和受体激酶。

该研究得到了Russian Foundation for Basic Research（批准号为1801600075）和国家VIR任务的（预算项目号为066220190006）的支持。

REFERENCES

1. Marchal E. De la specialisation du paristisme chez l'Erysiphe graminis. *Compt Rend Acad Sci Paris*. 1902;135:210-212.
2. Troch V, Audenaert K, Bekaert B, et al. Phylogeography and virulence structure of the powdery mildew population on its 'new' host triticale. *BMC Evol Biol*. 2012; 12:76. https://doi.org/10.1186/1471-2148-12-76.
3. Klocke B, Flath K, Miedaner T. Virulence phenotypes in powdery mildew (*Blumeria graminis*) populations and resistance genes in triticale (×*Triticosecale*). *Eur J Plant Pathol*. 2013;137(3):463-476. https://doi.org/10.1007/s10658-013-0257-9.
4. Hsam SL, Zeller FJ. Breeding for powdery mildew resistance in common wheat (*Triticum aestivum* L.). In: R.R. Belanger, W.R. Bushnell, A.J. Dik, T.L. Carver, ed. The Powdery Mildews, a Comprehensive Treatise. APS Press: St. Paul, Minnesota, USA; 2002. P. 219-238.
5. Flor HH. Current status of the gene-for-gene concept. *Annu Rev Phytopathol*. 1971;9(1):275-296. https://doi.org/10.1146/annurev.py.09.090171.001423.
6. Zhang Y, Lubberstedt T, Xu M. The genetic and molecular basis of plant resistance to pathogens. *J Genet Genomics*. 2013;40(1):23-35. https://doi.org/10.1016/j.jgg.2012.11.003.
7. Dodds PN, Rathjen JP. Plant immunity: towards an integrated view of plant-pathogen interactions. *Nat Rev Genet*. 2010;11(8):539-548. https://doi.org/10.1038/nrg2812.
8. Briggle LW. Near-isogenic lines of wheat with genes for resistance to *Erysiphe graminis* f. sp. *tritici*. *Crop Sci*. 1969;9(1):70-72. https://doi.org/10.2135/cropsci1969.0011183X000900010023x.
9. Briggle LW, Sears ER. Linkage of resistance to *Erysiphe graminis* f sp. *tritici* (*Pm3*) and hairy glume (*Hg*) on chromosome 1A of wheat. *Crop Sci*. 1966;6(6): 559-561. https://doi.org/10.2135/cropsci1966.0011183X000600060017x.
10. Zeller FJ, Lutz J, Stephan U. Chromosome location of genes for resistance to powdery mildew in common wheat (*Triticum aestivum* L.). 1. *Mlk* and other alleles at the *Pm3* locus. *Euphytica*. 1993;68(3): 223-229. https://doi.org/10.1007/BF00029876.
11. Sourdille P, Robe P, Tixier MH, et al. Location of Pm3g, a powdery mildew resistance allele in wheat, by using a monosomic analysis and by identifying associated molecular markers. *Euphytica*. 1999;110(3): 193-198. https://doi.org/10.1023/A:1003713219799.
12. Zeller FJ, Hsam SL. Progress in breeding for resistance to powdery mildew in common wheat (*Triticum aestivum* L.). In: A.E. Slinkard, ed. Proceedings of the 9th International Wheat Genetics Symposium; 1998 Aug 2-7; Saskatoon, SK, Canada. Saskatoon: University of Saskatchewan; 1998. P. 178-180.
13. Yahiaoui N, Kaur N, Keller B. Independent evolution of functional *Pm3* resistance genes in wild tetraploid wheat and domesticated bread wheat. *Plant J*. 2009;57(5):846-856. https://doi.org/10.1111/j.1365-313X.2008.03731.x.
14. Bhullar NK, Street K, Mackay M, et al. Unlocking wheat genetic resources for the molecular identification of previously undescribed functional alleles at the *Pm3* resistance locus. *Proc Natl Acad Sci USA*. 2009;106(23): 9519-9524. https://doi.org/10.1073/pnas.0904152106.
15. Shi AN, Leath S, Murphy JP. A major gene for powdery mildew resistance transferred to common wheat from wild einkorn wheat. *Phytopathology*. 1998;88(2): 144-7. https://doi.org/10.1094/PHYTO.1998.88.2.144.

16. Lowry JR, Sammons DJ, Baenziger PS, Moseman JG. Identification and characterization of the gene conditioning powdery mildew resistance in Amigo wheat. *Crop Sci*. 1984;24(1):129-132. https://doi.org/10.2135/cropsci1984.0011183X002400010030x.
17. Heun M, Friebe B, Bushuk W. Chromosomal location of the powdery mildew resistance gene of Amigo wheat. *Phytopathology*. 1990;80(10):1129-1133. https://doi.org/10.1094/Phyto-80-1129.
18. Hao YF, Liu AF, Wang YH, et al. Pm23: a new allele of *Pm4* located on chromosome 2AL in wheat. *Theor Appl Genet*. 2008;117(8):1205-1212. https://doi.org/10.1007/s00122-008-0827-y.
19. Li G, Cowger C, Wang X, et al. Characterization of *Pm65*, a new powdery mildew resistance gene on chromosome 2AL of a facultative wheat cultivar. *Theor Appl Genet*. 2019;132(9):22625-32. https://doi.org/10.1007/s00122-019-03377-2.
20. Mohler V, Bauer C, Schweizer G, et al. *Pm50*: a new powdery mildew resistance gene in common wheat derived from cultivated emmer. *J Appl Genet*. 2013;54(3):259-263. https://doi.org/10.1007/s13353-013-0158-9.
21. The TT, McIntosh RA, Bennett FG. Cytogenetical studies in wheat. IX. Monosomic analyses, telocentric mapping and linkage relationships of genes *Sr21*, *Pm4*, and *Mle*. *Aust J Biol Sci*. 1979;32(1):115-125. https://doi.org/10.1071/BI9790115.
22. Schmolke M, Mohler V, Hartl L, et al. A new powdery mildew resistance allele at the *Pm4* locus transferred from einkorn (*Triticum monococcum*). *Mol Breed*. 2012;29(2):449-456. https://doi.org/10.1007/s11032-011-9561-2.
23. Alam MA, Xue F, Wang C, Ji W. Powdery mildew resistance genes in wheat: identification and genetic analysis. *J Mol Biol Res*. 2011;1(1):20-39. https://doi.org/10.5539/jmbr.v1n1p20.
24. Sun H, Hu J, Song W, et al. Pm61: a recessive gene for resistance to powdery mildew in wheat landrace Xuxusanyuehuang identified by comparative genomics analysis. *Theor Appl Genet*. 2018;131(10):2085-2097. https://doi.org/10.1007/s00122-018-3135-1.
25. Reader SM, Miller TE. The introduction into bread wheat of a major gene for resistance to powdery mildew from wild emmer wheat. *Euphytica*.1991;53(1):57-60. https://doi.org/10.1007/BF00032033.
26. Zhang R, Sun B, Chen J, et al. *Pm55*, a developmental-stage and tissue-specific powdery mildew resistance gene introgressed from *Dasypyrum villosum* into common wheat. *Theor Appl Genet*. 2016;129(10):1975-1984. https://doi.org/10.1007/s00122-016-2753-8.
27. Hao M, Liu M, Luo J, et al. Introgression of powdery mildew resistance gene *Pm56* on rye chromosome arm 6RS into wheat. *Front Plant Sci*. 2018;9:1040. https://doi.org/10.3389/fpls.2018.01040.
28. Qi LL, Cao MS, Chen PD, et al. Identification, mapping, and application of polymorphic DNA associated with resistance gene *Pm21* of wheat. *Genome*. 1996;39(1):191-7. https://doi.org/10.1139/g96-025.
29. Hsam SL, Huang XQ, Earnst F, et al. Chromosomal location of genes for resistance to powdery mildew in common wheat (*Triticum aestivum* L. em Thell.). 5. Alleles at the *Pm1* locus. *Theor Appl Genet*. 1998;96(8):1129-1134. https://doi.org/10.1007/s001220050848.
30. Singrun CH, Hsam SL, Hartl L, et al. Powdery mildew resistance gene *Pm22* in cultivar Virest is a member of the complex *Pm1* locus in common wheat (*Triticum aestivum* L. em Thell.). *Theor Appl Genet*. 2003;106(8):1420-1424. https://doi.org/10.1007/s00122-002-1187-7.
31. Schneider DM, Heun M, Fischbeck G. Inheritance of the powdery mildew resistance gene *Pm9* in relation to *Pm1* and *Pm2* of wheat. *Plant Breed*. 1991;107(2):161-164. https://doi.org/10.1111/j.1439-0523.1991.tb00545.x.
32. Tan C, Li G, Cowger C, et al. Characterization of *Pm59*, a novel powdery mildew resistance gene in Afghanistan wheat landrace PI 181356. *Theor Appl Genet*. 2018;131(5):1145-1152. https://doi.org/10.1007/s00122-018-3067-9.
33. Perugini LD, Murphy JP, Marshall D, Brown-Guedira G. *Pm37*, a new broadly effective powdery mildew resistance gene from *Triticum timopheevii*. *Theor Appl Genet*. 2008;116(3):417-425. https://doi.org/10.1007/s00122-007-0679-x.
34. Zou SZ, Wang H, Li YW, et al. The NB-LRR gene *Pm60* confers powdery mildew resistance in wheat. *New Phytol*. 2018;218(1):298-309. https://doi.org/10.1111/nph.14964.
35. Peusha H, Enno T, Pruliin O. Chromosomal location of powdery mildew resistance genes and cytogenetic analysis of meiosis in common wheat cultivar Meri. *Hereditas*. 2000;132(1):29-34. https://doi.org/10.1111/j.1601-5223.2000.00029.x.

36. Lillemo M, Asalf B, Singh RP, et al. The adult plant rust resistance loci *Lr34/Yr18* and *Lr46/Yr29* are important determinants of partial resistance to powdery mildew in bread wheat line Saar. *Theor Appl Genet*. 2008;116(8):1155-1166. https://doi.org/10.1007/s00122-008-0743-1.
37. Hsam SL, Lapochkina IF, Zeller FJ. Chromosomal location of genes for resistance to powdery mildew in common wheat (*Triticum aestivum* L. em Thell.). 8. Gene *Pm32* in a wheat-Aegilops speltoides translocation line. *Euphytica*. 2003;133(3):367-370. https://doi.org/10.1023/A:1025738513638.
38. Lukaszewski AJ. Manipulation of the 1RS. 1BL translocation in wheat by induced homoeologous recombination. *Crop Sci*. 2000;40(1):216-225. https://doi.org/10.2135/cropsci2000.401216x.
39. Zhao Z, Sun H, Song W, et al. Genetic analysis and detection of the gene *MlLX99* on chromosome 2BL conferring resistance to powdery mildew in the wheat cultivar Liangxing 99. *Theor Appl Genet*. 2013;126(12):3081-3089. https://doi.org/10.1007/s00122-013-2194-6.
40. Tan C, Li G, Cowger C, et al. Characterization of *Pm63*, a powdery mildew resistance gene in Iranian landrace PI 628024. *Theor Appl Genet*. 2019;132(4):1137-44. https://doi.org/10.1007/s00122-018-3265-5.
41. Jørgensen JH, Jensen CJ. Gene *Pm6* for resistance to powdery mildew in wheat. *Euphytica*. 1973;22(2):423. https://doi.org/10.1007/BF00022656.
42. Rong JK, Millet E, Manisterski J, Feldman M. A new powdery mildew resistance gene: introgression from wild emmer into common wheat and RFLP-based mapping. *Euphytica*. 2000;115(2):121-126. https://doi.org/10.1023/A:1003950431049.
43. Zhu ZD, Zhou RG, Kong XY, et al. Microsatellite markers linked to 2 powdery mildew resistance genes introgressed from *Triticum carthlicum* accession PS5 into common wheat. *Genome*. 2005;48(4):585-590. https://doi.org/10.1139/G05-016.
44. Wei H, Liu ZJ, Zhu J, et al. Identification and genetic mapping of *pm42*, a new recessive wheat powdery mildew resistance gene derived from wild emmer (*Triticum turgidum* var. *dicoccoides*). *Theor Appl Genet*. 2009;119(2):223-230. https://doi.org/10.1007/s00122-009-1031-4.
45. Piarulli L, Gadaleta A, Mangini G, et al. Molecular identification of a new powdery mildew resistance gene on chromosome 2BS from *Triticum turgidum* ssp. *dicoccum*. *Plant Sci*. 2012;196:101-106. https://doi.org/10.1016/j.plantsci.2012.07.015.
46. Zhang D, Zhu K, Dong L, et al. Wheat powdery mildew resistance gene *Pm64* derived from wild emmer (*Triticum turgidum* var. *dicoccoides*) is tightly linked in repulsion with stripe rust resistance gene *Yr5*. *Crop J*. 2019;7(6):761-770. https://doi.org/10.1016/j.cj.2019.03.003.
47. Liu W, Koo DH, Xia Q, et al. Homoeologous recombination based transfer and molecular cytogenetic mapping of powdery mildew resistant gene *Pm57* from *Aegilops searsii* into wheat. *Theor Appl Gene*t. 2017;130(4):841-848. https://doi.org/10.1007/s00122-017-2855-y.
48. Zhan H, Li G, Zhang X, et al. Chromosomal location and comparative genomics analysis of powdery mildew resistance gene *Pm51* in a putative wheat — *Thinopyrum ponticum* introgression line. *PLoS One*. 2014;9(11): e113455. https://doi.org/10.1371/journal.pone.0113455.
49. Zhang R, Fan Y, Kong L, et al. *Pm62*, an adult-plant powdery mildew resistance gene introgressed from *Dasypyrum villosum* chromosome arm 2VL into wheat. *Theor Appl Genet*. 2018;131(12):2613-20. https://doi.org/10.1007/s00122-018-3176-5.
50. Li G, Fang T, Zhang H, et al. Molecular identification of a new powdery mildew resistance gene *Pm41* on chromosome 3BL derived from wild emmer (*Triticum turgidum* var. *dicoccoides*). *Theor Appl Genet*. 2009;119(3):531-539. https://doi.org/10.1007/s00122-009-1061-y.
51. Cenci A, D'Ovidio R, Tanzarella OA, et al. Identification of molecular markers linked to *Pm13*, an *Aegilops longissima* gene conferring resistance to powdery mildew in wheat. *Theor Appl Genet*. 1999;98(3-4):448-454. https://doi.org/10.1007/s001220051090.
52. Friebe B, Jiang J, Raupp WJ, et al. Characterization of wheat-alien translocations conferring resistance to diseases and pests: current status. *Euphytic*a. 1996;91(1):59-87. https://doi.org/10.1007/BF00035277.
53. Liu ZY, Sun QX, Ni ZF, et al. Molecular characterization of a novel powdery mildew resistance gene *Pm30* in wheat originating from wild emmer. *Euphytica*. 2002;123(1):21-29. https://doi.org/10.1023/A:1014471113511.
54. Blanco A, Gadaleta A, Cenci A, et al. Molecular mapping of the novel powdery mildew resistance gene *Pm36* introgressed from *Triticum turgidum*

var. *dicoccoides* in durum wheat. *Theor Appl Genet*. 2008;117(1):135-142. https://doi.org/10.1007/s00122-008-0760-0.

55. Petersen S, Lyerly JH, Worthington ML, et al. Mapping of powdery mildew resistance gene *Pm53* introgressed from *Aegilops speltoides* into soft red winter wheat. *Theor Appl Genet*. 2015;128(2):303-312. https://doi.org/10.1007/s00122-014-2430-8.
56. Tosa Y, Tokunaga H, Ogura H. Identification of a gene for resistance to wheatgrass powdery mildew fungus in common wheat cultivar Chinese Spring. *Genome*. 1988;30(4):612-14. https://doi.org/10.1139/g88-103.
57. Tosa Y, Sakai K. The genetics of resistance of hexaploid wheat to the wheatgrass powdery mildew fungus. *Genome*. 1990;33(2):225-230. https://doi.org/10.1139/g90-035.
58. Hao Y, Parks R, Cowger C, et al. Molecular characterization of a new powdery mildew resistance gene *Pm54* in soft red winter wheat. *Theor Appl Genet*. 2015;128(3):465-476. https://doi.org/10.1007/s00122-014-2445-1.
59. Järve K, Peusha HO, Tsymbalova J, et al. Chromosomal location of a *Triticum timopheevii*: derived powdery mildew resistance gene transferred to common wheat. *Genome*. 2000;43(2):377-381. https://doi.org/10.1139/g99-141.
60. Jia J, Devos KM, Chao S, et al. RFLP-based maps of homoeologous group-6 chromosomes of wheat and their application in the tagging of *Pm12*, a powdery mildew resistance gene transferred from *Aegilops speltoides* to wheat. *Theor Appl Genet*. 1996;92(5):559-565. https://doi.org/10.1007/BF00224558.
61. Friebe B, Heun M, Tuleen N, et al. Cytogenetically monitored transfer of powdery mildew resistance from rye into wheat. *Crop Sci*. 1994;34(3):621-625. https://doi.org/10.2135/cropsci1994.0011183X003400030003x.
62. Hsam SL, Huang XQ, Zeller FJ. Chromosomal location of genes for resistance to powdery mildew in common wheat (*Triticum aestivum* L. em. Thell.) 6. Alleles at the *Pm5* locus. *Theor Appl Genet*. 2001;102(1):127-133. https://doi.org/10.1007/s001220051627.
63. Huang XQ, Wang LX, Xu MX, Roder MS. Microsatellite mapping of the powdery mildew resistance gene *Pm5e* in common wheat (*Triticum aestivum* L.). *Theor Appl Genet*. 2003;106(5):858-865. https://doi.org/10.1007/s00122-002-1146-3.
64. Xiao M, Song F, Jiao J, et al. Identification of the gene *Pm47* on chromosome 7BS conferring resistance to powdery mildew in the Chinese wheat landrace Hongyanglazi. *Theor Appl Genet*. 2013;126(5):1397-1403. https://doi.org/10.1007/s00122-013-2060-6.
65. Luo PG, Luo HY, Chang ZJ, et al. Characterization and chromosomal location of *Pm40* in common wheat: a new gene for resistance to powdery mildew derived from *Elytrigia intermedium*. *Theor Appl Genet*. 2009;118(6):1059-1064. https://doi.org/10.1007/s00122-009-0962-0.
66. Tosa Y, Tsujimoto H, Ogura H. A gene involved in the resistance of wheat to wheatgrass powdery mildew fungus. *Genome*. 1987;29(6):850-852. https://doi.org/10.1139/g87-145.
67. Huang XQ, Hsam SL, Zeller FJ, et al. Molecular mapping of the wheat powdery mildew resistance gene *Pm24* and marker validation for molecular breeding. *Theor Appl Genet*. 2000;101(3):407-14. https://doi.org/10.1007/s001220051497.
68. Huang XQ, Röder MS. High-density genetic and physical bin mapping of wheat chromosome 1D reveals that the powdery mildew resistance gene *Pm24* is located in a highly recombinogenic region. *Genetica*. 2011;139(9):1179-1187. https://doi.org/10.1007/s10709-011-9620-y.
69. Xue F, Wang C, Li C, et al. Molecular mapping of a powdery mildew resistance gene in common wheat landrace Baihulu and its allelism with *Pm24*. *Theor Appl Genet*. 2012;125(7):1425-1432. https://doi.org/10.1007/s00122-012-1923-6.
70. Wiersma AT, Pulman JA, Brown LK, et al. Identification of *Pm58* from *Aegilops tauschii*. *Theor Appl Genet*. 2017;130(6):1123-1133. https://doi.org/10.1007/s00122-017-2874-8.
71. He R, Chang Z, Yang Z, et al. Inheritance and mapping of powdery mildew resistance gene *Pm43* introgressed from *Thinopyrum intermedium* into wheat. *Theor Appl Genet*. 2009;118(6):1173-1180. https://doi.org/10.1007/s00122-009-0971-z.
72. Herrera-Foessel SA, Singh RP, Lillemo M, et al. *Lr67/Yr46* confers adult plant resistance to stem rust and powdery mildew in wheat. *Theor Appl Genet*. 2014;127(4):781-789. https://doi.org/10.1007/s00122-013-2256-9.
73. Xu H, Yi Y, Ma P, et al. Molecular tagging of a new broad-spectrum powdery mildew resistance allele *Pm2* in Chinese wheat landrace Niaomai. *Theor*

Appl Genet. 2015;128(10):2077-2084. https://doi.org/10.1007/s00122-015-2568-z.

74. Gao H, Zhu F, Jiang Y, et al. Genetic analysis and molecular mapping of a new powdery mildew resistant gene *Pm46* in common wheat. *Theor Appl Genet*. 2012;125(5):967-973. https://doi.org/10.1007/s00122-012-1886-7.
75. McIntosh RA, Dubcovsky J, Rogers WJ, et al. Catalogue of gene symbols for wheat: 2013-2014 supplement. Available from: https://shigen.nig.ac.jp/wheat/komugi/genes/macgene/supplement2013.pdf.
76. Qiu Y, Sun X, Zhou R, et al. Identification of microsatellite markers linked to powdery mildew resistance gene *Pm2* in wheat. *Cereal Res Commun*. 2006;34(4):1267-1273. https://doi.org/10.1556/CRC.34.2006.4.268.
77. Miranda LM, Murphy JP, Marshall D, Leath S. *Pm34*: a new powdery mildew resistance gene transferred from *Aegilops tauschii* Coss. to common wheat (*Triticum aestivum* L.). *Theor Appl Genet*. 2006;113(8):1497-1504. https://doi.org/10.1007/s00122-006-0397-9.
78. Miranda LM, Murphy JP, Marshall D, et al. Chromosomal location of *Pm35*, a novel *Aegilops tauschii* derived powdery mildew resistance gene introgressed into common wheat (*Triticum aestivum* L.). *Theor Appl Genet*. 2007;114(8):1451-6. https://doi.org/10.1007/s00122-007-0530-4.
79. Ma PT, Xu HX, Xu YF, et al. Molecular mapping of a new powdery mildew resistance gene *Pm2b* in Chinese breeding line KM2939. *Theor Appl Genet*. 2015;128(4):613-622. https://doi.org/10.1007/s00122-015-2457-5.
80. Ma H, Kong Z, Fu B, et al. Identification and mapping of a new powdery mildew resistance gene on chromosome 6D of common wheat. *Theor Appl Genet*. 2011;123(7):1099-1106. https://doi.org/10.1007/s00122-011-1651-3.
81. Spielmeyer W, McIntosh RA, Kolmer J, Lagudah ES. Powdery mildew resistance and *Lr34/Yr18* genes for durable resistance to leaf and stripe rust cosegregate at a locus on the short arm of chromosome 7D of wheat. *Theor Appl Genet*. 2005;111(4):731-735. https://doi.org/10.1007/s00122-005-2058-9.
82. Lutz J, Hsam SL, Limpert E, Zeller FJ. Chromosomal location of powdery mildew resistance genes in *Triticum aestivum* L. (common wheat). 2. Genes *Pm2* and *Pm19* from *Aegilops squarrosa* L. *Heredity*. 1995;74(2):152-156. https://doi.org/10.1038/hdy.1995.22.
83. Zeller FJ, Kong L, Hartl L, et al. Chromosomal location of genes for resistance to powdery mildew in common wheat (*Triticum aestivum* L. em Thell.). 7. Gene *Pm29* in line Pova. *Euphytic*a. 2002;123(2):187-194. https://doi.org/10.1023/A:1014944619304.
84. Li Z, Lan C, He Z, et al. Overview and application of QTL for adult plant resistance to leaf rust and powdery mildew in wheat. *Crop Sci*. 2014;54(5):1907-1925. https://doi.org/10.2135/cropsci2014.02.0162.
85. Tang S, Hu Y, Zhong S, Luo P. The potential role of powdery mildew-resistance gene *Pm40* in Chinese wheat-breeding programs in the post-*Pm21* era. *Engineering*. 2018;4:500-506. https://doi.org/10.1016/j.eng.2018.06.004.
86. Švec M, Miklovičová M. Structure of populations of wheat powdery mildew *(Erysiphe graminis* DC f. sp. *tritici* Marchal) in Central Europe in 1993-1996: I. Dynamics of virulence. *Eur J Plant Pathol*. 1998;104(6):537-544. https://doi.org/10.1023/A:1008642816326.
87. Hurni S, Brunner S, Stirnweis D, et al. Powdery mildew resistance gene *Pm8* derived from rye is suppressed by its wheat ortholog *Pm3*. *Plant J*. 2014;79(6):904-913. https://doi.org/10.1111/tpj.12593.
88. Jørgensen JH, Wolfe M. Genetics of powdery mildew resistance in barley. *CRC Crit Rev Plant Sci*. 1994;13(1):97-119. https://doi.org/10.1080/07352689409701910.
89. Wei F, Gobelman-Werner K, Morroll SM, et al. The Mla (powdery mildew) resistance cluster is associated with three NBS-LRR gene families and suppressed recombination within a 240-kb DNA interval on chromosome 5S (1HS) of barley. *Genetics*. 1999;153(4):1929-1948.
90. Seeholzer S, Tsuchimatsu T, Jordan T, et al. Diversity at the *Mla* powdery mildew resistance locus from cultivated barley reveals sites of positive selection. *Mol Plant Microbe Interact*. 2010;23(4):497-509. https://doi.org/10.1094/MPMI-23-4-0497.
91. Kusch S, Panstruga R. *mlo*-Based resistance: an apparently universal "weapon" to defeat powdery mildew disease. *Mol Plant Microbe Interact*. 2017;30(3):179-189. https://doi.org/10.1094/MPMI-12-16-0255-CR.

92. Dreiseitl A. Genes for resistance to powdery mildew in European barley cultivars registered in the Czech Republic from 2011 to 2015. *Plant Breeding*. 2017;136(3): 351-356. https://doi.org/10.1111/pbr.12471.
93. Ociepa T, Okoń S, Nucia A, et al. Molecular identifcation and chromosomal localization of new powdery mildew resistance gene *Pm11* in oat. *Theor Appl Genet*. 2020;133(1):179-185. https://doi.org/10.1007/s00122-019-03449-3.
94. Hsam SL, Mohler V, Zeller FJ. The genetics of resistance to powdery mildew in cultivated oats (*Avena sativa* L.): current status of major genes. *J Appl Genetics*. 2014;55(2):155-162. https://doi.org/10.1007/s13353-014-0196-y.
95. Hsam SL, Zeller FJ. Chromosomal location of genes for resistance to powdery mildew in cultivated oat (*Avena sativa* L.). 1. Gene *Eg-3* in the cultivar Mostyn. *Plant Breed*. 1998;117(2):177-178. https://doi.org/10.1111/j.1439-0523.1998.tb01474.x.
96. Mohler V, Zeller FJ, Hsam SL. Molecular mapping of powdery mildew resistance gene *Eg-3* in cultivated oat (*Avena sativa* L. cv. 'Rollo'). *J Appl Genetics*. 2012;53(2):145-148. https://doi.org/10.1007/s13353-011-0077-6.
97. Yu J, Herrmann M. Inheritance and mapping of a powdery mildew resistance gene introgressed from *Avena macrostachya* in cultivated oat. *Theor Appl Genet*. 2006;113(3):429-437. https://doi.org/10.1007/s00122-006-0308-0.
98. Oliver RE, Tinker NA., Lazo GR, et al. SNP discovery and chromosome anchoring provide the first physically-anchored hexaploid oat map and reveal synteny with model species. *PLoS ONE*. 2013;8(3): e58068. https://doi.org/10.1371/journal.pone.0058068.
99. Herrmann MH, Mohler V. Locating two novel genes for resistance to powdery mildew from *Avena byzantina* in the oat genome. *Plant Breed*. 2018;137(6): 832-838. https://doi.org/10.1111/pbr.12655.
100. Hsam SL, Peters N., Paderina EV, et al. Genetic studies of powdery mildew resistance in common oat (*Avena sativa* L.) I. Cultivars and breeding lines grown in Western Europe and North America. *Euphytica*. 1997;96(3):421-427. https://doi.org/10.1023/A:1003057505151.
101. Hsam SL, Paderina EV, Gordei S, Zeller FJ. Genetic studies of powdery mildew resistance in cultivated oat (*Avena sativa* L.). II. Cultivars and breeding lines grown in Northern and Eastern Europe. *Hereditas*. 1998;129(3):227-230. https://doi.org/10.1111/j.1601-5223.1998.00227.x.
102. Okoń S. Identifcation of powdery mildew resistance genes in Polish common oat (*Avena sativa* L.) cultivars using host-pathogen tests. *Acta Agrobot*. 2012;65(3): 63-68. https://doi.org/10.5586/aa.2012.008.
103. Okoń S. Effectiveness of resistant genes to powdery mildew in oat. *Crop Prot*. 2015;74:48-50. https://doi.org/10.1016/j.cropro.2015.04.004.
104. Okoń S, Ociepa T, Nucia A. Molecular identification of *Pm4* powdery mildew resistant gene in oat. *Not Bot Horti Agrobot Cluj Napoca*. 2018;46(2): 350-355. https://doi.org/10.15835/nbha46210904.
105. Okoń S, Paczos-Grzęda E, Ociepa T, et al. *Avena sterilis* L. genotypes as a potential source of resistance to oat powdery mildew. *Plant Dis*. 2016;100(10): 2145-2151. https://doi.org/10.1094/PDIS-11-15-1365-RE.
106. Okoń S, Ociepa T. Effectiveness of new sources of resistance against oat powdery mildew identified in *A. sterilis*. *J Plant Dis Prot*. 2018;125(5):505-510. https://doi.org/10.1007/s41348-018-0171-7.
107. Okoń SM, Chrząstek M, Kowalczyk K, Koroluk A. Identification of new sources of resistance to powdery mildew in oat. *Eur J Plant Pathol*. 2014;139(1):9-12. https://doi.org/10.1007/s10658-013-0367-4.
108. Okoń S, Ociepa T, Paczos-Grzęda E, Ladizinsky G. Evaluation of resistance to *Blumeria graminis* (DC.) f. sp. *avenae*, in *Avena murphyi* and *A. magna* genotypes. *Crop Protect*. 2018;106:177-181. https://doi.org/10.1016/j.cropro.2017.12.025.
109. Sánchez-Martín J, Rubiales D, Prats E. Resistance to powdery mildew (*Blumeria graminis* f. sp. *avenae*) in oat seedlings and adult plants. *Plant Pathol*. 2011;60(5):846-856. https://doi.org/10.1111/j.1365-3059.2011.02453.x.
110. Dodds PN, Lawrence GJ, Catanzariti AM, et al. Direct protein interaction underlies gene-for-gene specificity and coevolution of the flax resistance genes and flax rust avirulence genes. *Proc Natl Acad Sci U S A*. 2006;103(23):8888-8893. https://doi.org/10.1073/pnas.0602577103.
111. Ortiz D, de Guillen K, Cesari S, et al. Recognition of the *Magnaporthe oryzae* effector AVR-Piaby the decoy domain of the rice NLR immune receptor RGA5. *Plant Cell*. 2017;29(1):156-168. https://doi.org/10.1105/tpc.16.00435.

112. Vakhrusheva OA, Nedospasov SA. System of innate immunity in plants. *Mol Biol*. 2011;45(1):16-23. https://doi.org/10.1134/S0026893311010146.
113. Krattinger SG, Keller B. Molecular genetics and evolution of disease resistance in cereals. *New Phytol*. 2016;212(2):320-332. https://doi.org/10.1111/nph.14097.
114. Keller B, Wicker T, Krattinger SG. Advances in wheat and pathogen genomics: implications for disease control. *Annu Rev Phytopathol*. 2018;56(1):67-87. https://doi.org/10.1146/annurev-phyto-080516-035419.
115. Lu X, Kracher B, Saur IM, et al. Allelic barley MLA immune receptors recognize sequence-unrelated avirulence effectors of the powdery mildew pathogen. *Proc Natl Acad Sci USA*. 2016;113(42): E6486-E6495. https://doi.org/10.1073/pnas.1612947113.
116. Srichumpa P, Brunner S, Keller B, Yahiaoui N. Allelic series of four powdery mildew resistance genes at the *Pm3* locus in hexaploid bread wheat. *Plant Physiology*. 2005;139(2):885-895. https://doi.org/10.1104/pp.105.062406.
117. Bhullar NK, Zhang Z, Wicker T, Keller B. Wheat gene bank accessions as a source of new alleles of the powdery mildew resistance gene *Pm3*: a large scale allele mining project. *BMC Plant Biol*. 2010;10(1):88. https://doi.org/10.1186/1471-2229-10-88.
118. Wicker T, Oberhaensli S, Parlange F, et al. The wheat powdery mildew genome shows the unique evolution of an obligate biotroph. *Nat Genet*. 2013;45(9): 1092-1096. https://doi.org/10.1038/ng.2704.
119. Smirnova O, Shumny VK, Kochetov AV. Gene network and database for genes of wheat's resistance to pathogenic fungi. *Russ J Plant Physiol*. 2018;65(3):319-332. https://doi.org/10.1134/S1021443718030076.
120. Sánchez-Martín J, Steuernagel B, Ghosh S, et al. Rapid gene isolation in barley and wheat by mutant chromosome sequencing. *Genome Biol*. 2016;17(1):221. https://doi.org/10.1186/s13059-016-1082-1.
121. Yahiaoui N, Srichumpa P, Dudler R, Keller B. Genome analysis at different ploidy levels allows cloning of the powdery mildew resistance gene *Pm3b* from hexaploid wheat. *Plant J*. 2004;37(4):528-538. https://doi.org/10.1046/j.1365-313X.2003.01977.x.
122. Hurni S, Brunner S, Buchmann G, et al. Rye *Pm8* and wheat *Pm3* are orthologous genes and show evolutionary conservation of resistance function against powdery mildew. *Plant J*. 2013;76(6):957-969. https://doi.org/10.1111/tpj.12345.
123. Cao A, Xing L, Wang X, et al. Serine/threonine kinase gene *Stpk-V*, a key member of powdery mildew resistance gene *Pm21*, confers powdery mildew resistance in wheat. *Proc Natl Acad Sci USA*. 2011;108(19):7727-7732. https://doi.org/10.1073/pnas.1016981108.
124. Krattinger SG, Lagudah ES, Spielmeyer W, et al. A putative ABC transporter confers durable resistance to multiple fungal pathogens in wheat. *Science*. 2009;323(5919):1360-1363. https://doi.org/10.1126/science.1166453.
125. Moore JW, Herrera-Foessel S, Lan C, et al. A recently evolved hexose transporter variant confers resistance to multiple pathogens in wheat. *Nat Genet*. 2015;47(2):1494-1498. https://doi.org/10.1038/ng.3439.
126. Jordan T, Seeholzer S, Schwizer S, et al. The wheat *Mla* homologue *TmMla1* exhibits an evolutionarily conserved function against powdery mildew in both wheat and barley. *Plant J*. 2011;65(4):610-621. https://doi.org/10.1111/j.1365-313X.2010.04445.x.
127. Wan P, Ling L, Cao S, et al Isolation, chromosomal location, and expression analysis of putative powdery mildew resistance genes in wheat (*Triticum aestivum* L.). *Euphytica*. 2007;155(1):125-133. https://doi.org/10.1007/s10681-006-9313-2.
128. Wei F, Wing RA, Wise RP. Genome dynamics and evolution of the *Mla* (powdery mildew) resistance locus in barley. *Plant Cell*. 2002;14(8):1903-1917. https://doi.org/10.1105/tpc.002238.
129. Buschges R, Hollricher K, Panstruga R, et al. The barley *Mlo* gene: a novel control element of plant pathogen resistance. *Cell*. 1997;88(5):695-705. https://doi.org/10.1016/S0092-8674(00)81912-1.
130. Piffanelli P, Zhou F, Casais C, et al. The barley MLO modulator of defense and cell death is responsive to biotic and abiotic stress stimuli. *Plant Physiol*. 2002;129(3):1076-1085. https://doi.org/10.1104/pp.010954.
131. Piffanelli P, Ramsay L, Waugh R, et al. A barley cultivation-associated polymorphism conveys resistance to powdery mildew. *Nature*. 2004;430(7002): 887-891. https://doi.org/10.1038/nature02781.
132. Ge X, Deng W, Lee ZZ, et al. Tempered *mlo* broad-spectrum resistance to barley powdery mil-

dew in an Ethiopian landrace. *Sci Rep*. 2016;6(1): 29558. https://doi.org/10.1038/srep29558.

133. Deppe JP, Rabbat R, Hörtensteiner S, et al. The wheat ABC transporter Lr34 modifies the lipid environment at the plasma membrane. *J Biol Chem*. 2018;293(48):18667-18679. https://doi.org/10.1074/jbc.RA118.002532.
134. Milne RJ, Dibley KE, Schnippenkoetter W, et al. The wheat *Lr67* gene from the sugar transport protein 13 family confers multipathogen resistance in barley. *Plant Physiol*. 2019;179(4):1285-97. https://doi.org/10.1104/pp.18.00945.
135. Huckelhoven R, Fodor J, Preis C, Kogel KH. Hypersensitive cell death and papilla formation in barley attacked by the powdery mildew fungus are associated with hydrogen peroxide but not with salicylic acid accumulation. *Plant Physiol*. 1999;119(4):1251-1260. https://doi.org/10.1104/pp.119.4.1251.
136. Shen QH, Zhou F, Bieri S, et al. Recognition specificity and RAR1/SGT1 dependence in barley *Mla* disease resistance genes to the powdery mildew fungus. *Plant Cell*. 2003;15(3): 732-744. https://doi.org/10.1105/tpc.009258.
137. Maekawa T, Kracher B, Saur IM, et al. Subfamily-specific specialization of RGH1/MLA immune receptors in wild barley. *Mol Plant Microbe Interact*. 2019;32(1):107-119. https://doi.org/10.1094/MPMI-07-18-0186-FI.
138. Andersen EJ, Ali S, Reese RN, et al. Diversity and evolution of disease resistance genes in barley (*Hordeum vulgare* L.). *Evol Bioinform Online*. 2016; 12:99-108. https://doi.org/10.4137/EBO.S38085.
139. Liu J, Cheng X, Liu D, et al. The miR9863 family regulates distinct *Mla* alleles in barley to attenuate NLR receptor-triggered disease resistance and cell-death signaling. *PLoS Genet*. 2014;10(12): e1004755. https://doi.org/10.1371/journal.pgen.1004755.
140. Mahadevappa M, De Scenzo RA, Wise RP. Recombination of alleles conferring specific resistance to powdery mildew at the *Mla* locus in barley. *Genome*. 1994;37(3):460-468. https://doi.org/10.1139/g94-064.
141. Periyannan S, Moore J, Ayliffe M, et al. The gene *Sr33*, an ortholog of barley *Mla* genes, encodes resistance to wheat stem rust race Ug99. *Science*. 2013;341(6147):786-788. https://doi.org/10.1126/science.1239028.
142. Mago R, Tabe L, Vautrin S, et al. Major haplotype divergence including multiple germin-like protein genes, at the wheat *Sr2* adult plant stem rust resistance locus. *BMC Plant Biol*. 2014;14(1):379. https://doi.org/10.1186/s12870-014-0379-z.
143. Jørgensen IH. Discovery, characterization and exploitation of Mlo powdery mildew resistance in barley. *Euphytica*. 1992;63(1-2):141-152. https://doi.org/10.1007/BF00023919.
144. Kim MC, Panstruga R, Elliott C, et al. Calmodulin interacts with MLO to regulate defence against mildew in barley. *Nature*. 2002;416(6879): 447-450. https://doi.org/10.1038/416447a.
145. Freialdenhoven A, Peterhansel C, Kurth J, et al. Identification of genes required for the function of non-race-specific *mlo* resistance to powdery mildew in barley. *Plant Cell*. 1996;8:5-14. https://doi.org/10.1105/tpc.8.1.5.
146. Wolter M, Hollricher K, Salamini F, Schulze-Lefert P. The *mlo* resistance alleles to powdery mildew infection in barley trigger a developmentally controlled defence mimic phenotype. *Mol Gen Genet*. 1993;239 (1-2):122-128. https://doi.org/10.1007/bf00281610.
147. Kokina A, Legzdina L, Berzina I, et al. Molecular marker-based characterization of barley powdery mildew MLO resistance locus in European varieties and breeding lines. *Agronomijas Vestis* (*Latvian Journal of Agronomy*). 2008;11:77-82.
148. Acevedo-Garcia J, Kusch S, Panstruga R. Magic mistery tour: MLO proteins in plant immunity and beyond. *New Phytol*. 2014;204(2):273-281. https://doi.org/10.1111/nph.12889.
149. Алпатьева Н.В., Абдуллаев Р.А., Анисимова И.Н., и др. Устойчивые к мучнистой росе образцы местного ячменя из Эфиопии // Труды по прикладной ботанике, генетике и селекции. – 2016. – Т. 177. – № 4. – С. 70–78. [Alpatyeva NV, Abdullaev RA, Anisimova IN, et al. Local barley accessions from Ethiopia resistant to powdery mildew. *Works on applied botany, genetics and plant breeding*. 2016;177(4):70-78. (In Russ.)]. https://doi.org/10.30901/2227-8834-2016-4-70-78.
150. Абдуллаев Р.А., Алпатьева Н.В., Яковлева О.В., и др. Генетическое разнообразие образцов ячменя из Эфиопии по устойчивости к мучнистой росе // Российская сельскохозяйственная наука. – 2019. – № 2. – С. 7–10. [Abdullaev RA, Alpatyeva NV, Yakovleva OV, et al. Genetic diversity of barley accessions from Ethiopia for the

powdery mildew resistance. *Russian Agricultural Science*. 2019;(2):7-10. (In Russ.)]. https://doi.org/10.31857/S2500-2627201927-10.

151. Elliott C, Zhou F, Spielmeyer W, et al. Functional conservation of wheat and rice *Mlo* orthologs in defense modulation to the powdery Mildew fungus. *Mol Plant Microbe Interact*. 2002;159(10):1069-1077. https://doi.org/10.1094/MPMI.2002.15.10.1069.
152. Nguyen VN, Vo KT, Park H, et al. A systematic view of the *MLO* family in rice suggests their novel roles in morphological development, diurnal responses, the light-signaling pathway, and various stress responses. *Front Plant Sci*. 2016;7:1413. https://doi.org/10.3389/fpls.2016.01413.
153. Ablazov A, Tombuloglu H. Genome-wide identification of the mildew resistance locus O (*MLO*) gene family in novel cereal model species *Brachypodium distachyon*. *Eur J Plant Pathol*. 2015;145(2):239-53. https://doi.org/10.1007/s10658-015-0833-2.
154. Appiano M, Catalano D, Martínez MS, et al. Monocot and dicot MLO powdery mildew susceptibility factors are functionally conserved in spite of the evolution of class-specific molecular features. *BMC Plant Biol*. 2015;15(1):257. https://doi.org/10.1186/s12870-015-0639-6.
155. Acevedo-Garcia J, Spencer D, Thieron H, et al. *mlo*-based powdery mildew resistance in hexaploid bread wheat generated by a non-transgenic TILLING approach. *Plant Biotechnol J*. 2017;15(3):367-378. https://doi.org/10.1111/pbi.12631.
156. Reinstadler A, Muller J, Czembor J, et al. Novel induced *mlo* mutant alleles in combination with site-directed mutagenesis reveal functionally important domains in the heptahelical barley Mlo protein. *BMC Plant Biol*. 2010;10(1):31. https://doi.org/10.1186/1471-2229-10-31.
157. Ingvardsen CR, Massange-Sánchez JA, Borum F, et al. Development of *mlo*-based resistance in tetraploid wheat against wheat powdery mildew. *Theor Appl Genet*. 2019;132(11):3009-22. https://doi.org/10.1007/s00122-019-03402-4.
158. Zhang Y, Bai Y, Wu G, et al. Simultaneous modification of three homoeologs of *TaEDR1* by genome editing enhances powdery mildew resistance in wheat. *Plant J*. 2017;91(4):714-724. https://doi.org/10.1111/tpj.13599.
159. Вавилов Н.И. Законы естественного иммунитета растений к инфекционным заболеваниям. (Ключи к нахождению иммунных форм) // Вавилов Н.И. Избранные труды. Т. 4. – М.; Л.: Наука, 1964. – С. 430–488. [Vavilov NI. Zakony yestestvennogo immuniteta rasteniy k infektsionnym zabolevaniyam. Klyuchi k nakhozhdeniyu immunnykh form. In: Vavilov NI. Izbrannye trudy. Vol. 4. Moscow; Leningrad: Nauka; 1964. P. 430-488. (In Russ.)]
160. Wolfe MS, McDermott JM. Population genetics of plant pathogen interactions: the example of the *Erysiphe graminis — Hordeum vulgare* pathosystem. *Annu Rev Phytopathol*. 1994;32(1):89-113. https://doi.org/10.1146/annurev.py.32.090194.000513.

❀ Author and affiliations

Eugeny E. Radchenko – Doctor of Science, Main Researcher, Department of Genetics. Federal Research Center "N.I. Vavilov All-Russian Institute of Plant Genetic Resources", St. Petersburg, Russia. SPIN: 1667-0530. E-mail: eugene_radchenko@rambler.ru.

Renat A. Abdullaev – PhD, Researcher, Department of Genetics. Federal Research Center "N.I. Vavilov All-Russian Institute of Plant Genetic Resources", St. Petersburg, Russia. E-mail: abdullaev.1988@list.ru.

Irina N. Anisimova – Doctor of Science, Leader Researcher, Department of Genetics. Federal Research Center "N.I. Vavilov All-Russian Institute of Plant Genetic Resources", St. Petersburg, Russia. SPIN: 5000-3256. E-mail: irina_anisimova@inbox.ru.